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DISEASES OF THE NERVOUS SYSTEM

A PRACTICAL JOURNAL ON PSYCHIATRY AND NEUROLOGY

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DISEASES of the NERVOUS SYSTEM

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A PRACTICAL JOURNAL ON PSYCHIATRY AND NEUROLOGY

Friedreich's Ataxia

(A Familial Hereditary Study of Three Cases)*

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Friedreich's ataxia, one of the many unusual heredofamilial degenerative neurological diseases, is rare but of great interest. The onset is usually early in life occurring between the ages of 5 and 14 years, with a few cases according to Woofter and Parks[1] reported as late as 39 to 50 years. The course of the disease is invariably progressive without remission except in the few reported cases of an abortive or arrested form.

The disease was first described by Friedreich[2] in 1861. He reported a primary hereditary disease of the cord with the outstanding pathological lesions confined primarily to the posterior and lateral columns. In the original article Friedreich described the hereditary disease as causing slow progressive ataxia of the lower and then of the upper extremities. He emphasized the absence of sensory disturbances or loss of sphincter control. His description of the disease and pathological findings have withstood the test of time for accuracy. However, his theory that the disease was closely related to tabes dorsalis was definitely proven incorrect in later years.

The disease is definitely hereditary as well as familial as illustrated in our genelogical chart Fig. 1. It can invariably be traced back for several generations and occurs in both males and females. The age onset is about the same in members of the same generation. The mode of transmission is obscure being transmitted by both male and female members of afflicted families. Some authorities postulate two morbid factors one of which is dominant and one recessive. Unquestionably, there is transmitted in some families a common genetic vulnerability of certain tracts and organs in the nervous system, the degeneration of which may occur in simple or complex fashion and which may produce simple or complicated neurological disturbances. In the first group we have the well delineated and true to form Friedreich ataxia while in the more complex group we have combination with other neurological manifestation such as the combination of Charcot-Marie-Tooth Atrophy and Friedreich's ataxia, etc., as reported by Ross[3] and Brugsch.[4]

In a survey of the literature we have selected only those cases reported which were believed to have all the necessary cardinal features of Friedreich's ataxia. We have avoided the inclusion of the complex groups such as the combinations or the cases which have insufficient data to warrant the true diagnosis of Friedreich's ataxia. Table I is a presentation of 28 cases which, in our estimation, are true classical cases of Friedreich's ataxia. Ataxia is found in 100 per cent of the cases described, and listed in the order of their maximum frequency of occurrence are

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the following additional findings, namely: deformity of the feet or pes cavus, deformity of the spine, loss of deep reflexes, nystagmus, positive Babinski sign, speech defect, impaired superficial sensation, impaired deep sensation, muscle atrophy and spina bifida.

The first symptom of stumbling while walking or running with the impaired locomotion is probably due to involvement of the propioceptive fibers of the posterior columns or the columns of Goll and Burdach. The various major and minor asynergias can be attributed to the involvement of the ascending cerebellar pathways and possibly of the cerebellum. According to Hassin[5] the nystagmus may be explained on lesions of the vestibulospinal pathways with secondary involvement of the vestibulo-cerebellar pathways. The degeneration of the lateral columns in all probability accounts for the positive Babinski sign and the various deformities of the feet and spine, namely, the pes cavus and kyphosis, lordosis and scoliosis of the spine. The degeneration of the propioceptive fibers beyond the sensory ganglia accounts for the loss of the tendon reflexes. The explosive type of speech disturbance is probably due to degeneration of other centripetal tract connections. There are no major sensory disturbances in any of the cases except of the propioceptive fibers. In the true cases of Friedreich's ataxia mentality is usually unimpaired.

As stated, the disease is usually progressive with increase in the muscle atrophy and skeletal deformities. In the terminal stages speech becomes incomprehensible and the patient becomes a total invalid and bedridden.

Figure 1: Genealogical chart of four generations. The affected members are represented by the black shading.

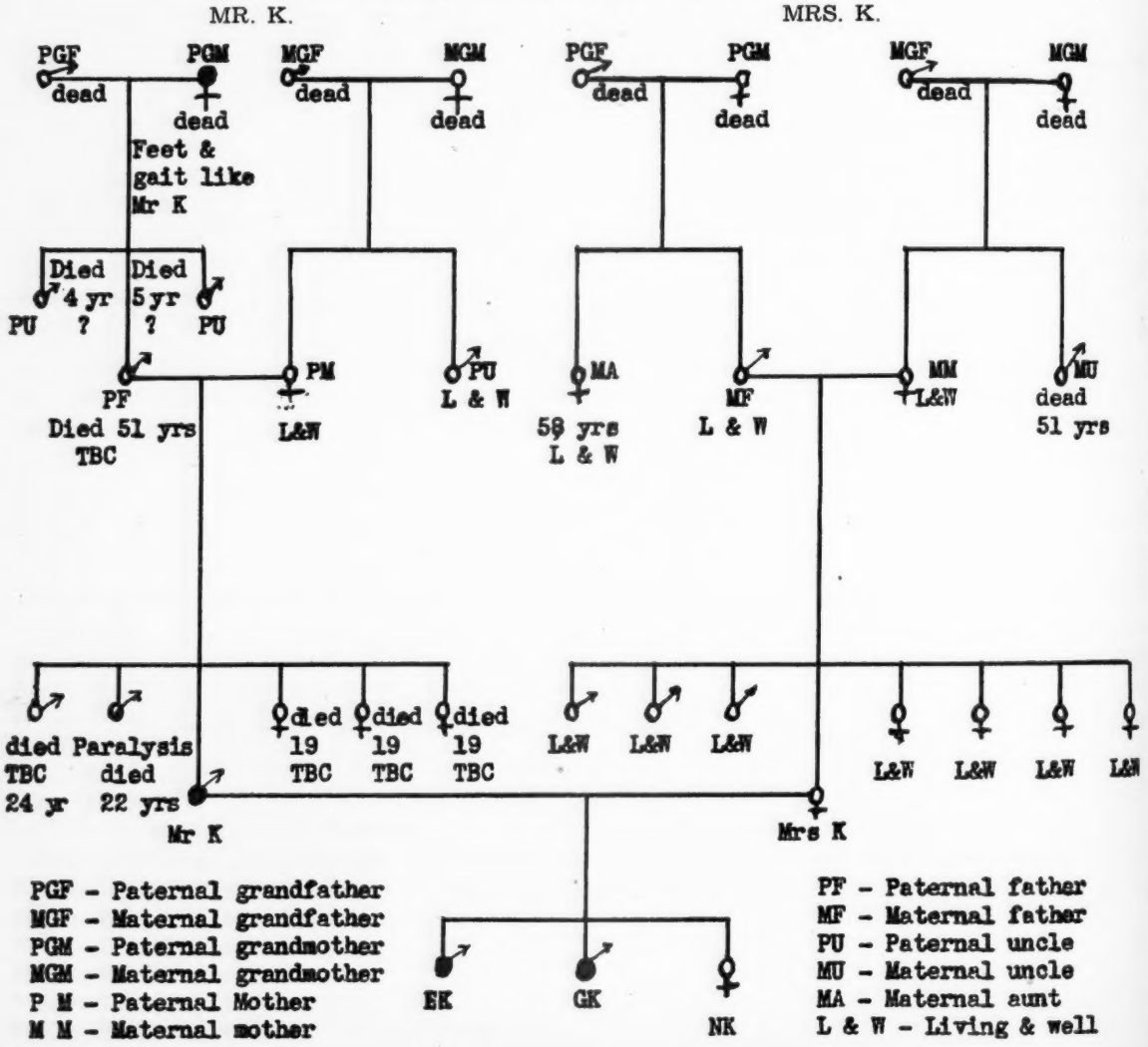

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TABLE 1

SUMMARY OF MAJOR FINDINGS IN 28 REPORTED CASES OF FRIEDREICH'S ATAXIA

Authors	Ataxia	Deformities of Spine	Deformities of Feet	Nystagmus	Loss of deep Reflexes	Babinski	Speech Defect	Impaired superficial Sensations	Impaired deep Sensations	Muscle Atrophy	Spina Bifida
Beer, G. 1.	x				x		x				
2.	x		x		x			x	x		
3.	x				x					x	
Bobonneix, et al 4.	x	x	x		x			x	x	x	
Alpers and Waggoner 5.	x	x	x	x	x	x		x			
6.	x	x	x		x		x			x	x
7.	x	x			x						x
8.	x	x			x						x
9.	x	x			x						x
10.	x	x			x						
Hiller 11.	x	x	x	x	x	x	x	x			
12.	x	x	x	x	x	x	x	x			
13.	x	x	x	x	x	x	x	x			
Popon 14.	x	x	x	x	x	x					
Rabinowitsch 15.	x	x					x	x			
Enziere 16.	x	x	x		x		x	x	x		
17.	x	x	x	x	x		x	x			
18.	x	x	x	x	x	x					
19.	x	x		x	x						
Orbon 20.	x	x	x	x	x	x					
21.	x	x	x	x	x	x	x	x	x		
22.	x	x	x	x	x	x					
Salus 23.	x	x	x	x	x	x	x	x			
Krebs 24.	x	x	x	x	x	x					
Pomme' et al 25.	x		x		x	x					
Lipson & DeNardi 26.	x	x	x	x	x	x	x	x		x	x
27.	x	x	x	x	x					x	x
28.	x	x	x	x	x					x	
SUMMARY	100%	85%	78%	57%	97%	50%	40%	43%	19%	22%	21%

The diagnosis is dependent upon the slowly progressive course, the positive heredo-familial history and the following outstanding findings:

(a) Defects typical of cerebellar dysfunction such as wide base, hypotonia, asthenia, poor equilibrium, nystagmus and speech disturbances.

(b) Characteristic deformities of feet and spine.

(c) Loss of deep tendon reflexes.

(d) Signs of involvement of the posterior columns of the spinal cord with loss of various propioceptive sensations, tabetic type of gait and positive Romberg sign.

(e) Signs of involvement of the pyramidal tracts, namely a positive Babinski.

Because several other diseases and certain toxic substances may cause changes in the posterior and lateral columns of the cord, they must be considered in the differential diagnosis. Pernicious anemia, leukemia, aplastic anemia, diabetes mellitus, pellagra, syphilis and chronic ergot poisoning may produce changes in the spinal columns but they are not primary conditions of the central nervous system and none produce the deformities of the feet and spine. Unlike Friedreich's ataxia multiple sclerosis is characterized by remissions, negative hereditary history and usual absence of deformities of the spine and feet. Amyotrophic lateral sclerosis may produce a pes cavus but the presence of muscle twitching and absence of any family history eliminates probable confusion with Friedreich's ataxia.

No specific treatment has been discovered for the arrest or retardation of the disease. The gait and stance of the individual may be improved by corrective orthopedic surgery of the feet and properly fitting footwear. Body braces and crutches are of some benefit as body support and as aid in locomotion especially in the late stages. In view of the unimpaired mentality of these cases every effort should be made for an educational program so that they may learn trades and professions befitting their progressive disability. A case of Friedreich's ataxia should be impressed with the hereditary factor and probable transmission of this factor through marriage.

The pathological anatomy has been thoroughly studied in several autopsies on cases of Friedreich's ataxia by both Spiller[6] and Hassin. Spiller believes that Friedreich's ataxia should not be considered a disorder confined to the spinal cord and cerebellum alone and suggests that if all investigators study their cases carefully they will find alterations not only in the cord and cerebellum but also in the peripheral nerves and muscles as well. In his autopsy findings he discovered that the heart was one-half the normal size, the posterior roots were observed to be much smaller in comparison to the anterior roots and that the spinal cord and medulla were grossly diminished in size. He found the absence of Clarke's column and their replacement by neuroglia. He also noted evidence of degeneration of the lateral pyramidal tracts and of the direct cerebellar tracts and their replacement by whorls of neuroglia.

Hassin in his histo-pathologic study of two cases of Friedreich's ataxia came to the conclusion that the pathologic feature of Friedreich's ataxia is degeneration of the postero-lateral columns of the spinal cord without involvement of the pons and cerebellum as are in Marie's ataxia. The outstanding histo-pathological picture presented by this author is the involvement of the posterior columns, Clarke's column and the posterior roots with preservation of the anterior roots, mild hyperplasia of the meninges and walls of the blood vessels and absense of signs of any inflammation. The lateral pyramidal tracts and the cerebellar tracts are involved later in the disease. He states that in his cases the antero-lateral tracts are not involved. In his opinion Friedreich's ataxia is primarily a disease of certain tracts of nerve fibers and any axonal reaction is secondary to retrograde degeneration of the tract fibers. He illustrates by stating that the axonal reactions in Clarke's columns are secondary to the long standing retrograde degeneration of the dorsal spino-cerebellar tract which originates in them.

Alpers and Waggoner[7] found evidence of extraneural and neural anomalies. They reported spina fibida to be present in 4 of their cases. We have discovered spina bifida occulta in two of our three cases.

In reporting our three cases, Fig. II, we have adhered to the standard of cardinal findings

for the accepted diagnosis of Friedreich's ataxia. There are two features in the symptomatology of our living cases which have been rarely noted or emphasized by other authors in the literature, namely: the excruciating pains in the thighs long before the appearance of the deformities and the neurological changes, and the frequent attacks of epistaxis being manifested during the same interval. The muscle pains in the thigh may be explained on the basis of propioceptive dysfunction. However, the epistaxis may have been a coincident or an unexplained haematological dysfunction associated with the disease.

In the study of the geneological chart, Fig. I, we discovered that the disease is transmitted through one generation from the grandmother to the grandson. It is also of importance to note that three sisters of the grandson and one brother died of pulmonary tuberculosis all in their youth while a second brother died of a "stroke" with paralysis at 22 years of age. The medical records on these cases are inadequate and it is quite possible that one or all of these members may have been afflicted with the heredo-familial disease in question.

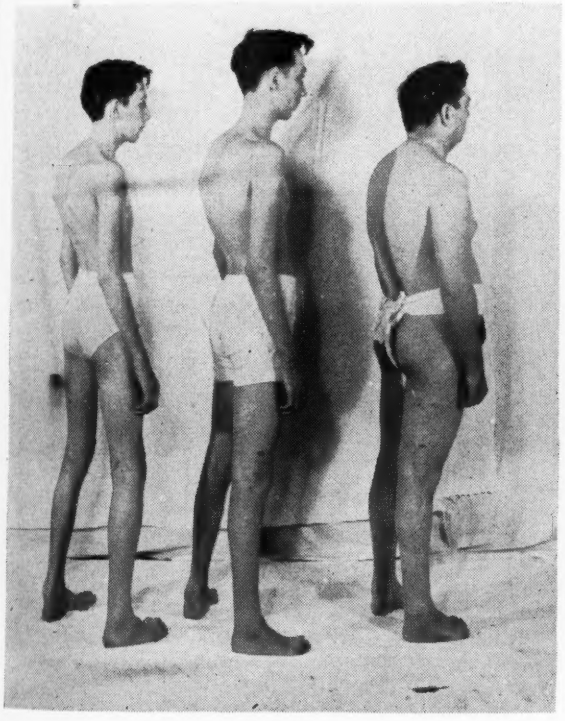

Figure II: The three reported living cases in our series, G.K., E.K. Jr., and E.K. Sr. The skeletal deformities with wide stance and muscle atrophy are quite in evidence.

In presenting the three case histories and findings in their geneological order we obtained the information (from E.K. Sr.) that the paternal grandmother died in old age of unknown cause. She had a gait and deformities of the feet (pes cavus and talipes equinus varus) very similar to her grandson (E.K. Sr). She undoubtedly was afflicted with the abortive type of the disease.

Case I in our group was E. K. Sr., born in Croatia, January 31, 1899, and came to America in 1907. Between the sixth and ninth years he had attacks of excruciating pain of both thighs and also attacks of epistaxis. The attacks would last about an hour and would always be manifested during the evening or at night. His playmates noticed his small arched feet when he was approximately nine years of age. At ten he became aware that he walked with a wide stance and on the outer surface of his shoes. He had corrective orthepedic surgery performed on both feet at Cleveland City Hospital in 1914. It was of interest to note in his admission examination in 1914 that the patellar, super-patellar and achilles reflexes were absent bilaterally. Since 1914 the patient has noticed no change in the objective or subjective symptoms.

He married in 1926 and has three children, two of whom display typical Friedreich's ataxia. The third sibling, a girl (N. K.), fourteen years of age is in excellent health and free of any sign of the disease. The wife has always enjoyed good health, and her ancestry, as revealed by the geneological chart, has been free of any hereditary disease.

The physical and neurological examination on November 18, 1945, reveal stooping of the shoulders, and a thoracic kyphosis and scoliosis. Pes cavus and talipes equinus varus were present bilaterally. Muscular atrophy and hypotonicity were especially noticeable in all extremities but the least pronounced of the three cases. The remainder of the physical examination was negative except for the neurological findings.

Neurologically all the deep reflexes were found to be absent while the superficial reflexes were normally active. Nystagmus was pronounced and present in all directions. The Romberg sign was positive and the Gordon Holmes tests were also positive but not as pronounced as in his sons. The plantar response could not be determined with accuracy due to orthopedic surgery of the feet. There was a definite ataxia and balance was maintained on a wide stance.

X-rays of the lumbo-sacral spine failed to reveal any abnormalities. All other laboratory tests were negative.

It is of interest to note that there have been no subjective or objective findings in this individual indicating progress of the disease since 1914. We may consider this case as arrested or as an abortive type.

Case II: E. K. Jr., born October 10, 1927, a spontaneous delivery. At six months he had a severe attack of pneumonia. Following this he had mumps and measles. Physical and mental growth was normal up to five years when he began having severe attacks of epistaxis which subsided after the eighth year. At five he began complaining of paroxysmal attacks of severe excruciating pain of the quadricep areas of both thighs. The attacks would last two to three hours, would invariably occur during the evening or at night and at times would awaken the patient from his sleep. Medical aid was summoned on several occasions but no definite organic diagnosis was made. The attacks of pain would occur at weekly intervals and the severity and duration was about the same up to nine years of age. Since then the attacks have been less severe, the duration unaltered and they occur four to five times a year.

At eleven years of age he began to walk with a "flop" and a wide gait. The patient noticed the deformities of the feet and that he was walking on the outer aspect of his shoes. At the same time he developed deformities of the spine and shoulders. Locomotion became so impaired in 1940 that he was forced to use crutches for support and to stabilize his gait. In the same year he had corrective surgery performed on both feet with definite improvement. The progression of the disease since has been slow in comparison with his younger brother.

E. K. Jr. has been an average scholar, has a rather pleasant personality and cooperated well during the various examinations. He was examined in our neurological clinic on October 12, 1944, at which time the diagnosis of Friedreich's ataxia was made. The physical examination revealed a thoracic kyphosis and scoliosis with lumbar lordosis. The shoulders exhibited a marked "rounding" and the chest had the typical "pigeon breast" deformity. Pes cavus and talipes equinus varus were present on both feet. The muscle atrophy was present throughout the body but not as pronounced as in his brother. The remainder of the physical examination failed to reveal any abnormalities.

The neurological examination revealed the absence of all deep reflexes while the cremasteric and abdominal reflexes were present and active. The Romberg and Gordon Holmes rebound tests were definitely positive. Nystagmus was present in both horizontal directions and also in the vertical direction. The pyramidal and extrapyramidal plantar responses could not be evaluated because of the peculiar malformation of the feet and toes. The optic discs revealed abnormally deep cupping. There was a marked ataxia and the patient walked with a wide gait. The x-rays of the lumbo-sacral spine shows a spina bifida occulta fig. III. The urinalysis was negative and no other laboratory studies were performed on this case.

Case III: G. K. was born May 7, 1930. No illness except whooping cough and measles in childhood. The history in this individual is identical with that of his brother. The attacks of pain of the anterior aspect of the thighs began when he was five years old and their intensity, duration and frequency were the same as in the case of E. K. Jr.

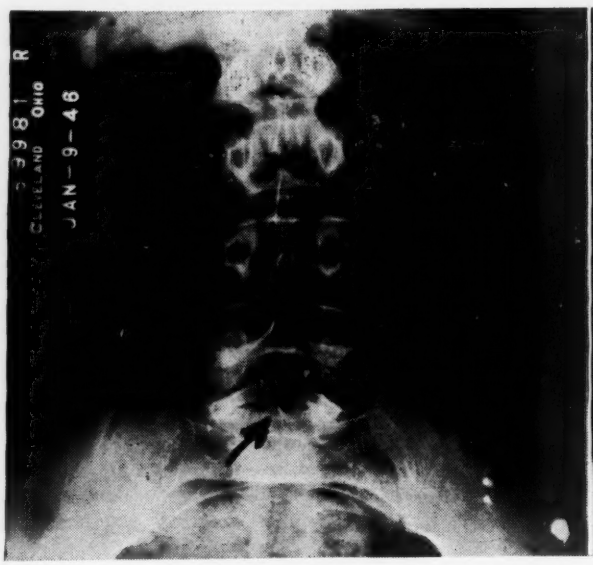

FIGURE III

FIGURE IV

X-rays of lumbo sacral spine revealing the spina bifida occulta in E.K. Jr. and G.K.

The attacks of epistaxis were frequently observed between the fifth and eighth years. At ten years of age the mother noticed the beginning of the "flopping" and "wide gait" in walking. She also noticed the "round shoulders" and beginning of spine deformities. All these deformities have been alarmingly more pronounced during the last year. He is able to walk only with great difficulty and there is no indication that the disease is abating as in the case of E. K. Jr. G. K. is an average student, has never failed and has a pleasant personality.

This patient was first seen in our orthopedic clinic on September 1, 1944, and was examined in the neurological department on September 14, at which time the diagnosis of Friedreich's ataxia was made. The physical examination revealed a marked scoliosis, lordosis and kyphosis of the entire spine. The chest was markedly deformed and presented a "pigeon chest" type of deformity. The shoulders were stooped forward and "rounded". There was a pronounced generalized muscle atrophy and hypotonia especially of girdle areas and the extremities. There was a typical pes cavus and talipes equinus varus of both feet, the outstanding and most constant abnormality found in Friedreich's ataxia. The thyroid, heart, and lungs were normal; abdomen normal.

The neurological findings were most outstanding in this case. The deep reflexes were all absent. The abdominal and cremasteric reflexes were normal to reaction. The Romberg sign was positive. The Gordon Holmes test was positive bilaterally. There was a marked ataxia on walking and a wide stance was necessary for balance. The optic discs revealed abnormally deep cupping. The plantar response could not be evaluated because of the peculiar deformities of the feet.

On November 22, 1945, this patient was admitted to the hospital and encephalograms were made which failed to reveal any cortical or cerebellar atrophy. The Kline on the blood and spinal fluid was negative. The gum mastic curve was flat and normal The spinal fluid revealed a 2-plus Pandy, plus-minus Ross-Jones and total protein of 15 mgm. per 100. The urine and complete blood work were within normal limits. The electro-encephalogram was negative and normal. X-rays of the lumbo-sacral spine revealed a spina bifida occulta (Fig. IV).

Summary

Three cases of typical Friedreich's ataxia in two generations of one family with one case apparently arrested while the others still show signs of progression are reported.

Twenty-eight cases have been reviewed from the literature and selected as typical entities of the disease eliminating any combination with other heredo-familial manifestations. In the symptomatology attention is called to the attacks of severe pain in the thighs and epistaxis long before the neurological changes and body deformities are manifested. Both symptoms have been rarely mentioned in the literature and their significance cannot be evaluated.

The diagnosis of the disease is dependent on the hereditary history, the ataxia including minor and major asynergias, the characteristic deformities of the feet and spine, the loss of deep tendon reflexes and the signs of involvement of the posterior columns and the lateral pyramidal tracts.

Two of the cases reported show x-ray evidence of spina bifida occulta which is an extraneural anomaly cited by others.

Treatment has been limited to corrective surgery and individual education to cope with the progression of the disease.

References

1 Woofter, A. C., and Parks, B. S.: "Friedreich's Ataxia," *Annals of Internal Medicine,* 17: 744, 1942.

2 Friedreich, N.: "Ueber Degenerative Atropiedes Spinalen Hinterstrange Virchow's," *Arch. Path. Anat.,* 26: 391, Berlin, 1863.

3 Ross, A. T.: "Combination of Friedreich's Ataxia and Charcot-Marie-Tooth Atrophy in two brothers," *J. of Nerv. and Ment. Dis.,* 95: 680, 1942.

4 Brugsch, H. G., and Hauptman, A.: "Friedreich's Ataxia with Charcot-Marie-Tooth's Neural Muscular Atrophy," *Bull. New Eng. Med. Center,* 6: 42, 1944.

5 Hassin, G. B.: "Friedreich's Ataxia. A Histopathologic Study of Two Cases," *Arch. of Neur. and Psych.,* 39: 116, 1938.

6 Spiller, W. G.: "Friedreich's Ataxia," *J. of Nerv. and Ment. Dis.,* 37: 411, 1910.

7 Alpers, B. J., and Waggoner, R. W.: "Extraneural and Neural Anomalies in Friedreich Ataxia," *Arch. of Neur. and Psych.,* 21: 47, 1929.

The Prevention of Maladjustment in an Army Service Forces Training Center

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The primary purpose of this paper is to present a viewpoint and *modus operandi* concerning the prevention of certain types of maladjustment observed early in an Army Service Forces Training Center at the Personnel Consultation Service by the neuropsychiatric consultant. Freedman[1,2,3,4] and others have discussed the organization and function of these mental hygiene clinics in training centers.

Of what value is the Psychiatrist in a Training Center? What is the specific scope of his activity and sphere of influence exclusive of the more general psychiatric functions?

I. *The Functions of the ASFTC Neuropsychiatrist*

There are five main functions:

1. Aiding the line officer in the stimulation of group adjustment and in the proper understanding of the soldiers' problems.
2. Prevention, which is most important and which is linked with early treatment and early recognition of those who are partially fit or the unfit.
3. Diagnosis—(Study).
4. Treatment—(Correction).
5. Disposition of the severely maladjusted and the unfit and/or prognostic evaluation.

II. *What General Types of Maladjustment or Incapacity Does the ASFTC Neuropsychiatrist Observe in His Initial Interview?*

From the standpoint of the relationship between the line officer and the Personnel Consultation Service, an important aspect of the Army Service Forces Training Center Neuropsychiatrist's first problem is recognition of difficulty with which he has to deal. There is a multiplicity of factors involved in the general psychiatric study of maladjustment. There are two general groupings, corresponding roughly with administrative or psychiatric determination, within which the ASFTC Personnel Consultation Service, at the initial interview, separate the patients referred by line officers or dispensaries:

(1) Ineffectuality which is ordinarily the psychiatrist's problem;
(2) Misconduct which usually falls into the province of the line officer.

This differentiation is not absolute and is subject to exceptions. However, as a first step in the *modus operandi,* cases can be placed in these two broad classifications. Moreover, there are other factors requiring future psychiatric study and further research. This is only an initial classification pending the gathering of all facts in regard to the cases.

Ineffectuality indicates that the individual does not adequately carry out useful activity. This may be temporary or permanent. The failure may be situational or on the basis of disability (physical or emotional), constitutional limitations (inaptitude or lack of adaptability), or because of poor motivation and possibly because of undesirable habits and character traits. This last type of ineffectuality would commonly come under the heading of misconduct.

Types of ineffectuality have been classified as mild, moderate and severe; they may be partial or total. The milder types are usually associated with situational problems such as a training situation and are usually acute, transitory and partial. The more severe reactions are relatively chronic and tend more often toward complete or full blown reactions although there are severe acute reactions. The severe cases are frequently associated with personality deviations rather than with the situation. One finds in the majority of the severe ineffectuals a poorly integrated personality structure. Intrapersonal conflicts, asocial attitude, or constitutional lack of adaptability are other factors to be considered in the study of these ineffectuals.

The psychiatrist's main function in this entire group is prevention and treatment. The emotions and unconscious intrapsychic conflicts which result in confusion and apathy are frequently the primary reaction tendency

and control the behavior of these individuals. They are very sensitive to group censure.

Misconduct on the other hand, is ordinarily an administrative or disciplinary problem. The individual engaging in misconduct does not adjust to the group nor accept its standards. As a result, he works against the welfare of the group. Misconduct may also be classified as mild, moderate or severe, partial or total, and temporary or permanent. The milder reactions are, as a rule, situational, accidental, or may be due to poor leadership or inadequate understanding of the soldier. The more severe cases, however, present aspects of prolonged social maladjustment and severe anti-social attitudes or behavior.

In this connection, "severe misconduct" is a term implying lack of moral and ethical values. It may arise from inherent limitations, anti-social attitude and at times from disability. If due to mental or emotional disability, this type of misconduct would be considered under ineffectuality.

Soldiers included in the category of severe misconduct are distinctly within the province of the line officer. These men are usually aggressive and work against the group. Their actions are anti-social. Their conflicts are directed against their fellow soldiers. There is conscious lack of control of their actions and purposeful disregard of group censure and social values.

From a long term psychiatric viewpoint, the severe cases of maladjustment may be considered to be incapacitated for military service. Incapacity, then in this sense, would mean that the individual is prevented from any effective functioning in the Army because of (1) disability, (2) lack of aptitude or inherent resources, (3) anti-social behavior which may, or may not, fall into the province of the psychiatrist.

Disability implies a pathologic condition which of itself incapacitates the individual and prevents him in varying degrees from normal functioning. It is beyond the individual's responsibility. It represents the results of injury or illness and is a definite medical problem.

Inaptitude or inadequacy implies an inherent lack of physical, intellectual or emotional stability which renders the individual "incapable" of effective military service.

However, disability and inaptitude (or constitutional inadequacy) may be considered under ineffectuality. Furthermore, it has been our experience that not infrequently, cases that appear to be inapt or inadequate during early interview, after careful study have been classified as cases of disability.

Another important factor with regard to misconduct and ineffectuality is the consideration whether the soldier is responsible for his acts. Those who evidence ineffectuality are usually not as responsible as those who engage in misconduct as many of the former often possess a mental or physical disability.

In the milder or earlier cases, it may be difficult to separate misconduct from ineffectuality because of the similarity of the basic mechanisms or the situations involved. Misconduct frequently has its roots in ineffectuality, i.e., the personal problems and the intrapsychic conflicts of the soldier or constitutional inadequacy or lack of plasticity. The training center psychiatrist can aid the line officer in understanding the situational factors involved, in understanding some of the reasons for the soldier's behavior, and in evaluating the personality of the individuals who engage in less serious misconduct. In this way he may aid in preventing the severe manifestations.

It is true that many factors must be considered by the psychiatrist including the soldier's reaction to the situation, the predominating group relationship and attitudes, previous integration of his personality, discipline, morals, etc. All the available factual data must be gathered and evaluated.

The psychiatrist's primary function, particularly in the more severe cases, is to decide whether the soldier engaging in misconduct is responsible or not. If he is not psychotic and is responsible, he is the line officer's problem. Psychiatry has a future function in connection with rehabilitation of severe cases of misconduct after disciplinary action is taken. However, for disposition at the time of his misconduct, the soldier primarily is the line officer's problem and only secondarily the problem of the ASFTC psychiatrist, and only in connection with the program of good leadership.

Everyone who shows ineffectuality or misconduct does not have to be discharged. Early

recognition and study of the difficulties the soldier may encounter may prevent the necessity of such action.

III. *Prevention of Maladjustment and the Stimulation of Group Adjustment*

A. *Preliminary Consideration.* Early recognition of the difficulties or problems which the soldier faces and a personal approach to the soldier is an important prerequisite to the application of preventive measures. Some of the factors involved in maladjustment and which respond to preventive measures are outlined below:

(a) Reaction to the training situation and change in mode of life in new trainees or harrowing experiences observed in overseas veterans.

(b) Home and family problems.

(c) Mild personal worries or anxiety evidenced by feelings of insecurity and worry over one's physical or mental condition or ability to "take it"—somatic complaints.

(d) Inter-personal and group relationships or attitudes, either obvious or hidden.

(e) Interplay between situation and type of personality.

(f) Inadequate leadership and poor discipline leading to poor morale.

(g) Mild personality mal-integration, mild degree of lack of plasticity or mildly restricted type of adaptability (usually acquired).

B. Briefly, in the prevention of maladjustment, the psychiatrist aids the commanding general in the concurrent planned promulgation of a definite program which includes:

1. Establishment of a general policy to aid in the stimulation of group adjustment and morale by means of specific recommendations; lectures to and conferences with officers, non-commissioned officers, and enlisted men; coordination of agencies concerned in handling the soldier, i.e., line officers, assignment section, dispensaries and hospital; and close linkage with the information and education branch, special service officer, chaplain, etc.

2. The treatment of soldier's individual problems. What procedure can be used in prevention of ineffectuality which may become severe or may lead to misconduct?

a. Early recognition of soldier's problem and early solution and immediate correction of the environmental factors which can be influenced.

b. Study of soldier's assets and liabilities (his personality structure).

c. Stimulation of constructive outlets (stimulation of his assets, proper assignment and advice in overcoming his liabilities).

d. Exploration of soldier's difficulties and explanation as to which of these he can correct, influence, utilize, or overcome, so that he can adjust to the group.

e. The aim of all psychotherapy should be to help a man stand on his own feet. The psychiatric service should be an agency which gives the man advice on how to help himself and where to obtain help if necessary.

Much of this therapy is indirect because it is shared by the line officers whom we aid in personalized handling and in evaluating the soldier. The leading questions which concern the line officer and the psychiatrist are:

1. What manner of man was he prior to induction? (Personality and family background).

2. What manner of man is he now? (Behavior, productions, actions in the group).

3. Why does he act the way he does?

4. What can the man do to help himself?

5. How can the Army or the group help him to help himself?

6. How can the team accept him and use him if he is able to accept the team—if he gives evidence of wanting to join the team?

7. Is the man engaging in purposeful misconduct or is he ineffectual?

Since the man who engages in misconduct generally does not accept the team, he is most often the line officer's problem. It is the commanding officer's function to provide incentives for him to mingle with the group, to join the team, to develop pride in his outfit, and to prevent excessive group censure, particularly if it is not warranted. He should never make an example of a man. All these considerations are linked with good discipline.

An attempt at teaching and education should go hand in hand with disciplinary measures. While the man, for the sake of the group, must learn to take the consequences of his acts, and to respect group opinion, punishment is not the primary motive. Education and utilization of the man and his assets are more important. Close linkage with

the information and educational branch is necessary to help in accomplishing this end.

The officer-psychiatrist, non-commissioned officer conference in regard to the individual case and their discussions of adequately prepared case abstracts is an important method in accomplishing prevention. Frequently, the soldier is invited to the conference after his case has been studied. His problems are discussed and a joint unified program of advice formulated.

As a result of these procedures, only about one per cent fail to adjust permanently. About ten per cent of the soldiers may have temporary or minor difficulties in adjustment from time to time but finally become adaptable to military service usually after reassignment to tasks which are commensurate with their specific adaptive capacities with a gradual induction into more difficult aspects of the training program.

Below are presented two cases illustrative of the points stressed:

Case of a Shrinking Individual Benefited by Treatment and Assignment

Family History:

This 21 year old soldier is the only child of a suburban middle class family. His father is 59 years old and handicapped by a spine injury. His mother is nervous, worrisome, and over-solicitous regarding the soldier. He has always been closely attached to his family, a home boy. He cries when he becomes nostalgic and thinks about home.

Previous Personal History:

The soldier completed high school at 18, after a fairly good academic adjustment but he had few friends at school, preferring to remain by himself. Following graduation he played the clarinet and saxophone in a band. He showed no anti-social tendencies. He did not make many friends, was seclusive, immature and closely attached to his home and his mother.

Military History:

The soldier was first seen by the psychiatrist during basic training when he was referred as an emergency case by his battalion commander after he passed out on the drill field and could not be brought to. The examiner found him lying on the ground in a mild stupor state from which he was easily aroused by pressure over the eyeballs. He arose quickly with little assistance.

Apparently this was a mild hysterical attack in a soldier frightened by the training situation and by the fact that he was told on the day previous that he would have to have an operation for a hernia. He feared that if he refused this operation he would be court-martialed and worried excessively. He claimed that the men in the company and his superiors shouted at him because he did things wrong and that he felt better if they didn't bother him because he couldn't stand being hollered at and took it too hard.

The soldier was returned to duty. The attack was construed as an attention getting mechanism associated with anxiety, but it was recommended that he be excused from arduous duties when he resumed training. These were to be again included after a short time.

A conference with the commanding officer elicited the information that after the soldier had been told that he must undergo the hernia operation he became extremely upset because he heard that someone had died as the result of such an operation. The soldier was afraid of both the operation and a possible court-martial for refusal. It was learned that the soldier had completed only a limited amount of training, claiming trouble with his feet; that he had spells of crying, at one time weeping constantly during three hours of rifle practice and at another time breaking down during the first firing period on the range because he claimed he was afraid to fire the rifle.

After this information was gathered, the soldier was returned to the Personnel Consultation Service for psychotherapy and help through suggestion. The commanding officer was instructed to make no mention of the episode, to observe the soldier without his being aware of it, to encourage him to do as much as he could but to excuse him if it appeared that he was going to drop out, and to instruct the men in the organization to pay as little attention as possible to the soldier and not to mention the fainting episode.

The soldier was seen many times at the Personnel Consultation Service and completed his training without further difficulty. Following this he was reassigned to the band where he performed adequately as a clarinetist.

An Aggressive Individual who went AWOL

Family History:

This 24 year old soldier was the fourth of nine children in an urban family of marginal economic status and meager socio-cultural background. Family relationships were quite loose and there was much friction between the soldier and the siblings and the soldier and his parents. The latter had been separated for ten years. The children were sickly and frail and several of his brothers had been rejected or been discharged after a short period of service. This soldier had been accustomed to having his own way and he shows definite rejection pattern to any discipline or authority at home. He had left home several times for no apparent reason except that

he wanted adventure on his own and finally left for good at the age of 16.

Previous Personal History:

He completed nine grades of school, leaving at the age of 15. The pattern of rejection of authority continued at school and there was some friction with his teachers. He was last employed as a pipe fitter for two years in a shipyard. Prior to that, he had been doing odd work as a painter, pipe fitter or plumber. His employment was sporadic and he did not get along well on his jobs. The soldier claimed a "shot-gun" wedding in 1939 and was divorced shortly thereafter. He remarried in 1943 and has a child. He was never well adjusted in civilian life. He was arrested in 1938 on a larceny charge but was acquitted. A year later he was arrested for burglary in Philadelphia and given a short sentence and placed on two years' probation. He always felt sorry for himself, blamed others for his difficulties, made excuses for his short-comings and tended to seek sympathy by magnifying minor physical ailments which he claimed interfered with his ability to work and to get along with people.

Military History:

The soldier was seen shortly after he arrived at the post to receive basic training and he claimed at first that he had moved his bowels only twice since induction. He later admitted that he did move his bowels but made many excuses, claiming the stools were not formed, his stomach pained him and that he could not adjust his personal habits to the speed with which the Army expected him to perform.

When he was next seen the soldier claimed that he had been absolutely well until he entered the Service and sought to blame all his difficulties on the Army. He continued to make excuses and tried to spare himself all blame. His company commander was contacted and the soldier was carefully observed for a period of time. One afternoon he became very upset and claimed that he could not take training and that nobody was doing anything for him. He was admitted to the Station Hospital.

He displayed a belligerent attitude in the hospital. He was seen by both medical and neuropsychiatric services and was returned to duty after a week's residence. The medical officer at the Station Hospital stated that he capitalized and exaggerated his complaints and that his motivation was poor.

A few weeks later in the training period he absented himself without leave for eighteen days after having written his wife several letters threatening to go AWOL because Army authorities were not doing anything for him and he could not take it any longer. He returned voluntarily. He resented discipline and thought he was being unfairly treated..

Later he stated that he could not sleep in the barracks and went out to sleep in the woods on a Saturday night. The soldier was again seen at this office and his excuse was that he did not think bed check was taken on Saturday night and he felt restless and walked to the woods where he fell asleep.

Despite his somatic complaints, the soldier presented a history of misconduct in civilian life and in the Service. He resented authority and was unable to conform to group life. Many of his actions are modified by his desire to avoid arduous duties and "to get out of the Army". He was irritable and surly. The status of temporary Limited Assignment was recommended. He received a reprimand and company punishment for the absence without leave.

After conference with us, his commanding officer took control of the situation. He talked to this trainee, explained again what was expected of him as a soldier, the necessity of doing as he was told and the importance of improving his motivation. The company commander worked with him for three months and under a firm but fair regime, despite his "griping," he began to improve in his adjustment. Later he was reassigned to Carpenters School in which work he said he was interested. He completed this course successfully and was assigned as a carpenter's helper. He was declared fit for overseas duty. He has been in no serious difficulty since.

Summary

1. Some of the functions of the neuropsychiatrist in a Personnel Consultation Service in an Army Service Forces Training Center with particular reference to assisting the line officer in the problem of adjustment of soldiers to group life in the Army have been indicated.

2. In this paper the importance and *modus operandi* of aiding the line officer in the stimulation of good adjustment and early recognition and treatment in the prevention of maladjustment have been stressed.

REFERENCES

1 Freedman, Harry L.: "The Services of the Military Mental Hygiene Unit," *Am. J. Psychiat.*, 100: 34-40, July 1943.

2 Cruvant, Bernard A.: "Replacement Training Center Consultation Service," *Am. J. Psychiat.*, 100: 41-46, July 1943.

3 TB Med. 156: "Consultation Service," *War Department Technical Bulletin*, June 1945.

4 Menninger, William C.: "Psychiatric Problems in the Army," *J. Amer. M. A.*, 123: 751-754, Nov. 20, 1943.

Plan of Organization of an Industrial Health Program

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Objectives

To plan a health program for any industrial organization it is necessary to first outline what is hoped to be accomplished. The primary objective of the program is to aid and encourage the efficient operation and functioning of the company it is to serve, with the end result of increasing production or services at lower costs. It can accomplish these aims if it improves the health, both mental and physical, of the employees and management; if it decreases absenteeism from work; if it helps to reduce the number of accidents; and if it thereby makes healthier, happier and more efficient employees.

Relationship to Industry

An industrial health program must fit, as a department, into the company organization. Its maximum value is dependent on its efficiency as a service department, and it can function properly only if the medical director is responsible to the top management—the president, the vice-president or the plant manager, as the case might be. The medical director must have an intimate knowledge of management policies, administrative procedures and the personnel of the organization, to efficiently plan and direct the work of his department. He must know the details of working conditions, potential toxic exposures and other health hazards.

Economics

In our system of free enterprise, a health program must justify itself as being good business for the company. A good health program is good business, as has been proven repeatedly by practically all major industries where careful economic controls are set up. It is good business in dollars and cents savings to the company, as a result of decreased absenteeism and increased efficiency of employees at work. In addition, there are many intangible values of a good health program for any company, large or small.

Minimum Standards

The American College of Surgeons has set up minimum standards for an industrial medical service which include adequate facilities, reputable physicians, complete records and general supervision of the sanitation of the plant and the health of the employees.

Medical Personnel

All medical personnel, physicians, nurses and other employees, must have all of the professional training and other requisites of success in private practice and the qualities which are necessary to fit into an industrial organization. There are many complicated problems which are a basic part of an industrial health program and which make it imperative to have the most capable doctors and nurses that can be secured. Some of the complexities of this type of practice are the proper handling of employees' personal and confidential health problems, as they are related to absenteeism, as they effect the work of others and in a manner to comply with benefit or insurance programs. The staff should be paid sufficiently high salaries to attract the best.

Specific Objectives of a Health Program

Employment Examinations: A complete physical examination, including chest x-ray, blood serology, and other laboratory work, is necessary for the intelligent placement and safe assignment of any employee.

Placement Examination: All employees should have a physical examination to include an evaluation of their emotional and psychological status, before they are assigned to a new job. Such information as the employee's relationship to his job and his fellow employees, and his personal history which can be obtained from his performance record, along with his medical history and physical

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examination, gives the industrial physician a complete picture and permits him to make such an evaluation. These criteria are also applied to old employees at the time of transfer to a new assignment.

Periodic Reexaminations: An industrial health program offers the best possible opportunity for routine periodic health examinations. These are accepted generally as the best means of preventive medicine available today. Such examinations should be complete and should be undertaken sympathetically, and with sufficient time to carefully evaluate the employee's health problems. A very large percentage of people have at least some psychosomatic problems. Quite remarkable results are seen in some cases, when, after complete physical examination, the employee is assured of the absence of organic disease and is acquainted with the significance of any symptoms he might have. Employees found to have medical problems may be followed at regular intervals: weekly, monthly, quarterly or yearly, as the case may be.

Special Examinations: In many industries special examinations are necessary on employees exposed to occupational disease hazards. This examination should first be made at the time the employee is assigned to the job and periodically thereafter, depending on the degree of exposure. The type and frequency of the examination in these cases varies with the particular job. Common examples of this procedure are employees exposed to lead, benzol, silica dust and other toxic materials.

In many communities equipment for periodic chest x-ray surveys is available through the local anti-tuberculosis organizations. These surveys are extremely beneficial in early case finding, and such services are usually accepted enthusiastically by employee groups.

Blood Tests: One of the accepted serologic tests for syphilis should be made at the time of each physical examination. The findings of these examinations should remain absolutely confidential between the physician and the employee. They should work out a program for the employee to receive treatment from his private physician, if treatment is necessary. I would like to emphasize again the necessity of not making the results of such tests a part of the employees' personnel records.

It is highly important for the physician who makes the periodic examinations to have a clear insight into the problems of mental hygiene, because in this field much can be done to contribute to the health, happiness and efficiency of employee groups.

Benefit Programs: Most companies have various benefit programs. These often constitute the payment of full salary or wages to employees losing time due to bona fide illness, depending on the length of service and increasing with the accumulation of service. Most major industrial companies today have group life and group hospitalization insurance programs, and many have retirement annuity plans for their employees. Such a health program as we are discussing should include specialized counseling to the company management in the planning and administration of these various programs, and to the employees to insure that they receive the full benefits to which they are entitled under the plans.

Dispensary: In industries having a sufficiently large number of employees, it is desirable to have a dispensary at the office or plant. All employees leaving work because of illness and those returning to work after illness should be checked by the doctor or nurse in attendance. This means is used to guide the employee toward proper care and treatment when he is leaving and to follow up his convalescense when he returns. Typhoid and smallpox vaccinations as well as other treatments and medication, on the direction and request of the employee's family physician, may be administered at the dispensary. The care and treatment of industrial injury cases is also handled there. Industries of sufficient size can efficiently utilize the services of a visiting nurse to maintain contact with employees who are off sick. The early treatment of upper respiratory infections is a valuable service.

Consultation Service: A medical department should be a place in the company where all employees should feel free to go for advice about any health problem. If the company is large enough, the department should include complete diagnostic facilities so that necessary examinations can be made in those

cases where indicated. Many large companies have complete x-ray equipment and a complete clinical laboratory where blood determinations of all types, urinalysis, smears and cultures may be made. It is well to include an electrocardiograph and a basal metabolism machine.

Returning Veterans: A definite program for returning veterans is essential. This should include a placement examination at the time of their return. Most companies have excellent programs for the intelligent and sympathetic handling of former employees returning from military service.

Sanitation: Standards should be established in cooperation with the management or the Engineering Department for safe water supply;f or sanitarya nd aesthetic wash rooms, and for bath room facilities in the plant. There should be sanitary standards for the camps where employees live.

Lighting, Heating and Ventilation: Minimum standards for lighting, heating and ventilation should be established.

Safety: There should be very close cooperation between the Medical Department, the Personnel Department and the Safety Department, since many problems encountered in industry are of joint responsibility. This is true in regard to the placement of workers with physical disability as well as those with psychological problems. These departments can cooperate in establishing safety rules and in the placing of responsibility for causes of accidents. I do not think it can be too strongly emphasized that some people are particularly susceptible to accidents and illness. Because of anxieties or worries, either on the job or at home; poor health habits, loss of sleep or fatigue; poor nutrition; or inherited constitutional weakness; these people are always in trouble of some kind. An appreciable percentage of accidents due to human failure is found to occur among this group. This is a joint problem in mental hygiene which concerns both management and the medical and safety departments.

Health Education: This important subject may be handled in many ways: at employee and management group meetings; by the distribution of pamphlets; and through news items in company publications. The best and most valuable means of health education is through the personal contact with the employee at the time of his periodic health examination.

Medical Records: Complete and accurate confidential medical records are essential. As previously stated, these should not be a part of the employee's regular company record. The information contained in such confidential records should be available only to the physician and the employee, and should not be used for any purpose other than to aid the employee in connection with company business. If such records are not confidential, the physician loses his basic relationship with the employee as a patient.

Handling of Accident Cases: It should be the policy of the company, its insurance carrier (if one exists), and the Medical Department, to see that all industrial injury cases receive the most competent medical attention available. These cases should be followed up after their return to work until they have fully recovered. The after care necessary to secure maximum improvement often lasts for weeks or months after the employee returns to work.

Relationship to the Medical Profession: A health program of this type should be so organized that it functions as a part of the essential medical service of the whole community. It should cooperate with the medical profession in the community and with the county and state health agencies. The services of the United States Public Health Service are available to all industries and are often a very valuable source of information.

Discussion and Resume

We have outlined the principles and specific objectives of an industrial health program. An organization must be styled to suit the particular needs of the industry concerned. This may vary from the part time services of a single physician to an intergraded medical department with many doctors and nurses, and elaborate hospital and diagnostic facilities. In general, such a program should anticipate the need of, and furnish advice to management and employees on all matters pertaining to the health of the employee. In any case, the principles are the same.

A Near Fatality from Curare Preceding Electro-Shock Therapy

Case Report

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Since 1939 the administration of curare immediately preceding electroshock and metrazol convulsive therapy has gained wide acceptance in the treatment of the various psychoses. Its purpose in this respect is to minimize the dangers of fractures occurring during the convulsive seizures. It is well known that curare has definite toxic properties, but when administered intravenously in doses calculated on the weight of the individual patient it is generally conceded to be a safe and therapeutically effective procedure. The purpose of this paper is to present a case in which the administration of curare preceding electroshock therapy was almost fatal.

Case Report

Mrs. E. S., a white female, age 26, presented herself with complaints of "drawing of the lips, difficulty in swallowing, and talking." The onset of sensation of "drawing of the upper lip" was in July 1944. This was especially noted in the right upper lip and rapidly increased in severity until she felt that she could no longer completely close her lips. Soon difficulty in swallowing food and marked impairment in precise pronounciation of words developed. These symptoms appeared in attacks, which at first usually occurred at night but several months later began to occur any time. They would become fully manifested within fifteen minutes from onset of the attack and would last from two to fifteen hours. It was noted that they were relieved by rest. These attacks continued until the time that she came to the hospital in October 1945. In addition the patient complained of generalized headache more severe in the occipital region and radiating to both shoulders. The headache was particularly painful during the attacks. She also had had recurring weakness of both upper extremities with numbness and tingling of her hands.

Her personal history revealed that the patient had been subject to periods during which she expressed illogical ideas of futility and had on occasions made threats of suicide all of which resembled a depressive pattern. She had always been a retiring, self-conscious individual subject to frequent spells of marked irritability.

Physical and neurological examinations were within normal limits. Routine laboratory studies were normal. X-ray of the lungs and heart was normal, and skull x-rays displayed no abnormalities. Electroencephalogram showed a normal pattern.

After several interviews it was concluded that there was an element of depression in this individual with a moderate amount of tension and anxiety symptoms. It was decided to give the patient a series of electroshock treatments in an effort to correct the depressive element of her illness.

When the patient reported for her first electroshock, she weighed 120 pounds. On the basis of this weight, she was given 45 milligrams curare administered intravenously at a rate that required sixty seconds for the full injection. Electroshock was never given because within one minute of the completion of the injection the patient had a mild generalized convulsive seizure, followed almost immediately by complete unconsciousness, rapid but shallow respirations and a moderate cyanosis. A second mild generalized convulsive seizure followed within two or three minutes, after which the patient showed a severe degree of cyanosis and very shallow respirations. The respiratory attempts thereafter became increasingly feeble and within ten minutes ceased altogether. The pulse became faint and the rate diminished to thirty per minute. Cyanosis was marked and the patient seemed about to expire. Shafer method of artificial respiration was instituted and pure oxygen was administered with the use of a mask and bag. One cc. of a 1:2000 solution of prostigmine, two cc. caffeine soda benzoate, one cc. coramine and one cc. of a 1:1000 solution of adrenaline were injected intravenously in rapid succession.

It became evident that there was an obstruction in the upper air passageway, and an artificial airway was placed in the patient's mouth with little improvement in respirations. The patient remained alive but failed to show any decrease in the severe degree of cyanosis or any return of respiratory function. Intravenous injection of one cc. prostigmine and one cc. coramine were repeated but still with no improvement. The obstruction in the upper air passageway appeared to be in the larynx. Consequently, the anesthesiology department was called upon to insert an intratrachial airway, aspirate the grad-

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ually increasing collection of mucus in the upper bronchial tree, and administer forced artificial respiration with an oxygen mask and bag.

Almost immediately the patient showed decrease in the cyanosis but the depth of unconsciousness continued, along with the weak, slow pulse and total lack of respiratory efforts. Intravenous injection of one cc. of 1:2000 solution of prostigmine was repeated every thirty minutes with no response. In spite of these efforts for over two hours it was thought that the patient would not survive. However, after the initial two hours the patient began to show gradual return of respiratory function and a favorable response to each intravenous injection of prostigmine. At the end of the third hour the intertrachial airway was removed and the remaining laryngeal paralysis was relieved with hourly intravenous administration of prostigmine.

The patient began to show return of consciousness after the end of the fourth hour. Six hours after the onset of this episode the only remaining evidence of this severe reaction to curare was a partial atelectasis of the lower lobe of the left lung and at that time the administration of prostigmine was discontinued. The atelectasis was relieved by frequent hyperventilation, by self-induced coughing and by sharply jarring the chest while the patient was holding her breath in deep inspiration. Twenty-four hours later the patient had made a complete recovery from the profound effects produced by the original intravenous injection of 45 milligrams of curare.

Discussion

No satisfactory explanation for this untoward reaction was ever established but several possibilities were considered. In order to rule out the possibility that the particular preparation of curare, of which the patient received the first and only injection, was not more potent than labeled, the entire supply of that particular control number of curare was returned to the manufacturers for reassaying. They reported that the drug involved was of the labeled potency and in addition was injected into a dog for continuous head-drop effect for a period of three hours with no abnormal reaction.

Another explanation for this massive severe response was the possibility that the patient had a previously unrecognized mild case of myasthenia gravis. Evidence that pointed to this diagnosis was the fact that the patient showed a favorable response to repeated injections of one cc. of a 1:2000 solution of prostigmine after the initial two hours of respiratory paralysis. But reviewing the history and physical findings we failed to be convinced that the diagnosis of mild myasthenia gravis was reasonable.

A third possibility was that this reaction could have been the result of anyphylactic shock response to the components of the curare extract, but since the patient had never before received an injection of curare, it was difficult to conceive how she could have been previously sensitized to this drug. Another argument against anyphylactic shock was the fact that the major features of this reaction were the respiratory paralysis and laryngeal obstruction. However, the initial convulsions and the copious outflow of bronchial secretion could be explained on this basis. It is possible that the reaction could have been anyphylactic in nature due to some foreign protein contained in the extract to which the patient had been previously sensitized by another drug.

A fourth explanation considered was that this patient was for some unknown reason unduly sensitive to ordinary safe doses of curare and that her response to 45 milligrams of curare was an exaggerated reaction in keeping with the pharmacological effects of the drug when administered in large doses. In order to prove this possibility it would be necessary to test the patient's individual sensitivity by the injection of minute doses of curare and increasing the dosage up to tolerance. In this instance such a procedure was not considered advisable merely for the sake of definitely establishing a diagnosis.

Conclusion

The value of curare in markedly reducing the incidence of fractures resulting from convulsive therapy has been proved conclusively through several years of clinical use. Curare, therefore, has made an undisputed place for itself in the convulsive treatment of the various psychoses. But at the same time it should be remembered that curare is a potentially dangerous drug with certain well known toxic actions and every possible precaution should be observed constantly during its use. The importance of having intravenous prostigmine, oxygen, artificial airways, intratracheal tubes, and central nervous system stimulants for immediate use cannot be overly stressed.

Neurological Clinical Pathological Conference

of the
Cincinnati General Hospital
CHARLES D. ARING, M.D., *Editor*

Case Number 131450

Presentation

The patient was a 34 year old colored man, who had been previously well. In October 1939 an ischio-rectal abscess was drained in this hospital. At that time the story and examination revealed nothing abnormal besides a systolic blood pressure ranging between 150 and 155, and a diastolic pressure between 85 and 100 mm. The admission temperature, pulse, and white blood count were in keeping with the ischio-rectal abscess. The single specimen of urine routinely examined had a specific gravity of 1.015 and there was a three plus test for albumin. Systemic recovery was prompt after drainage of the abscess.

The patient was next seen in a stuporous condition on the evening of June 21, 1945. His wife gave the following story: They had known about his high blood pressure for a bit over a year, and she said that he had always had heart trouble. The patient had been short of breath on exertion for about four months, and during that time he had had some spells in which he said he couldn't see. For about one month there had been some difficulty in passing urine, and slight swelling of the feet. For ten days he had complained of occipital and frontal headache. An hour and a half before his admission to the hospital he had "talked out of his head". He had had no bowel movement for about a week and had had an enema during the afternoon of June 21.

Admission examination on the evening of June 21 showed the following abnormalities: The patient was stuporous but he responded to pain and to his name; he was somewhat aware during the examination. The heart was enlarged to the left, and the apex beat was forceful. There was a harsh systolic murmur to the left of the sternum at the second interspace and in the pulmonic area. There was mild edema of the ankles. He hiccoughed occasionally. The respirations were fast and noisy.

There was slight side-to-side movement of the eyeballs, which was apparently involuntary. The eyes rotated to either canthus on passively rotating the head from side to side. Fine nystagmus was observed with the eyes deviated toward the right. The right palpebral fissure was wider than the left, and blinking did not close it as well as the left. The right upper eyelid resisted passive opening less well than the left. The optic discs were clearly outlined; the retinas were pale. There was extreme narrowing of the arteries and very full veins, and arteriovenous compression. Fresh hemorrhages were seen in the right retina. The corneal reflexes were equally weak. He responded to pinprick on the left face more quickly than to pinprick on the right face.

Resistance of the limbs to passive movement was reduced. On the evening of admission the patient was thought to have a left hemiparesis. On the following morning, all of his extremities fell equally quickly on testing them, but he moved the right lower extremity slightly better than the left when they were pricked with a pin. The abdominal reflexes were absent. There was a right Hoffmann response. There was no response to left plantar stimulation. The right plantar response was weakly extensor on 50 per cent of the trials; on the other 50 per cent there was a weak flexor response.

The patient remained stuporous, the edema increased and spread to involve the trunk and the face. "Uremic frost" was seen on the face just before death, which occurred early on the morning of June 24, after 66 hours of hospital stay.

Laboratory Data: The systolic blood pressure ranged between 240 and 280 (on occasions beyond the range of the manometer), the diastolic between 120 and 150. The blood analysis was as follows: hemoglobin 11 grams, red blood count 3.8 million, white blood count 19,500, the blood urea nitrogen 100 mg.%, blood Kahn negative. One urinalysis showed

a specific gravity of 1.010; and a three plus albumin test. There were 70 to 80 white blood cells and 4 to 5 red blood cells per high power field, with a few epithelial cells in the smear. A lumbar puncture done on June 22 revealed an initial pressure of 385 mm. of water. After the removal of 6 cc. of fluid resembling water, the final pressure was 190 mm. There were 4 white blood cells and 12 red blood cells per cmm. The protein was 130 mg.%, and the Wassermann reaction negative. There was a first zone gold curve (4442210000).

Differential Diagnosis

Dr. Eugene B. Ferris: The case is that of a 34 year old colored man with marked arterial hypertension presenting problems related to etiology and to the nature of the complications which resulted in death.

It is evident from the history and physical findings that we are dealing with malignant hypertension which progressed rapidly during the last few months of life. Though coma developed rapidly, it was possible to detect an initial crossed hemiplegia with sensory and motor involvement of the left extremities and the right side of the face. This suggests a lesion in the right pons involving the pyramidal tract and the facial and trigeminal nerves. Later the coma deepened and there were signs, such as a right Hoffmann and extensor plantar responses, suggesting diffuse lesions. In support of more diffuse cerebral involvement was the elevated cerebrospinal fluid pressure and protein content, as well as the deepening coma and the hemorrhage along the retinal veins of the right eye.

The high cerebrospinal fluid pressure and distended retinal veins indicate increased intracranial pressure; the high cerebrospinal fluid protein is evidence that the cerebral vessels were abnormally permeable to the passage of protein through their walls. The leukocytosis and the high cerebrospinal fluid protein probably represent in part a reaction to intracerebral hemorrhage. In view of the neurological signs which did not suggest a single large intracerebral hemorrhage, and the height of the cerebrospinal fluid pressure, I believe that in addition there was diffuse cerebral edema. These cerebral lesions are those to be expected in hypertensive encephalopathy. The visual disturbance which occurred over four months previously and the later headaches suggest that the vascular disorder of the brain had been active for some time.

Since the blood urea nitrogen in this case was over 100 mg.% and the patient was said to have developed uremic snow before death, the point is raised as to the role of the uremia in the production of the cerebral lesions and symptoms. This is a frequent consideration in cases of this sort because hypertension is a diffuse vascular disease and not infrequently in malignant hypertension as in this case, the blood vessels of the kidney and brain decompensate in parallel fashion. Uremia itself in the absence of cerebral vascular failure is not associated with hypertensive encephalopathy or focal vascular lesions of the brain, and its chief effect is one of increasing stupor or delirium. In this case, uremia must be of minor significance in the symptomatology because the abnormalities in consciousness and in neurological function were sudden in onset and rapid in development; the coma of uremia comes on slowly.

Before dismissing the uremia too casually, it should be pointed out that renal failure with developing uremia is associated with certain other systemic effects and it is not unusual for congestive heart failure, depression of hemopoesis and certain bleeding tendencies to occur. Whether it is the uremia per se or the widespread vascular change that often accompanies renal failure which accounts for these clinical effects is a debatable question, but it is quite likely that the renal insufficiency in this case may have accelerated the cerebrovascular disease, as it so often accelerates the cardiac failure in hypertensive cardiac disease.

Besides the interesting neurological features this case presents many other diagnostic problems which we meet commonly in hypertension. The first question which arises concerns the nature of the hypertension; is it essential or renal hypertension? There is some evidence in the clinical study to indicate that it is renal in origin. This conclusion is based primarily on the findings of "three plus" albuminuria when the patient was treated for ischio-rectal abscess in 1939; at that time the blood pressure was slightly elevated. This is a very important diagnostic

sign, for in essential hypertension with nephrosclerosis such a degree of albuminuria is extremely rare especially in the earlier phases of the disease. Further, there is evidence that the patient was developing progressive renal failure over the last few months of life as indicated by anemia, slight edema and difficulty in passing urine. Of course, mild congestive heart failure may have been present over this period; the widening of the heart border to the left and the harsh systolic murmur were indicative of left ventricular hypertrophy. However, nothing was said about congestive rales in the lungs or about attacks of nocturnal dyspnea or orthopnea which we would expect in hypertensive heart failure. Therefore, I am inclined to attribute the dyspnea and edema to slowly progressive renal failure.

The nature of the renal disease is more difficult to determine. We have already ruled out essential nephrosclerosis as the cause and there is nothing in the protocol to suggest acute glomerulonephritis in the past or to suggest other rare causes for hypertension such as periarteritis nodosa. It is unfortunate that the urinary sediment was not examined in 1939; however, on the final admission the urine contained many pus cells and because of this positive finding and lack of evidence of other renal diseases, I shall diagnose chronic pyelonephritis as the primary renal lesion.

It is a popular pastime to differentiate between benign and malignant hypertension which is of some importance in the earlier phases of hypertension for prognostic purposes. In this case we know the hypertensive vascular disease was malignant in nature for the cerebral lesions killed the patient at the age of 34, and there was considerable clinical evidence that the vascular disease was accelerated and malignant. In this regard, the point this patient brings out is that hypertension may be benign or malignant regardless of whether it is of the "essential" type or is due to specific renal disease.

My final diagnoses are: malignant hypertension, chronic pyelonephritis with renal insufficiency, and diffuse cerebral vascular disease with multiple small intracerebral hemorrhages ("ball" hemorrhages), and cerebral edema. Diffuse venous lesions, such as Doctor Scheinker has described in hypertensive encephalopathy, will also be seen.

Discussion of Pathology

Dr. I. Mark Scheinker: The extraneural findings were chronic pyelonephritis, nephrosclerosis, hypertrophy and dilatation of the heart, and generalized arteriosclerosis.

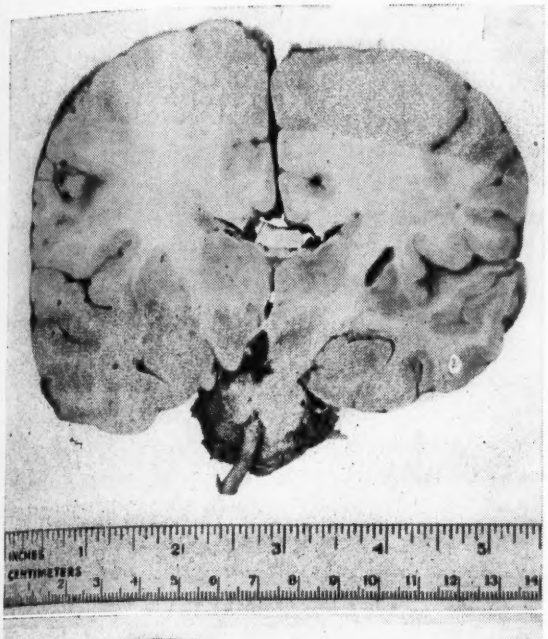
On gross examination of the brain the gyri were flattened and broadened, the sulci narrowed. There was an old area of softening about the posterior extent of the left Sylvian fissure. There was a well-defined cerebellar pressure cone. Although the temporal lobes appeared generally swollen, there was no uncal herniation. There was sclerosis of the vessels of the circle of Willis.

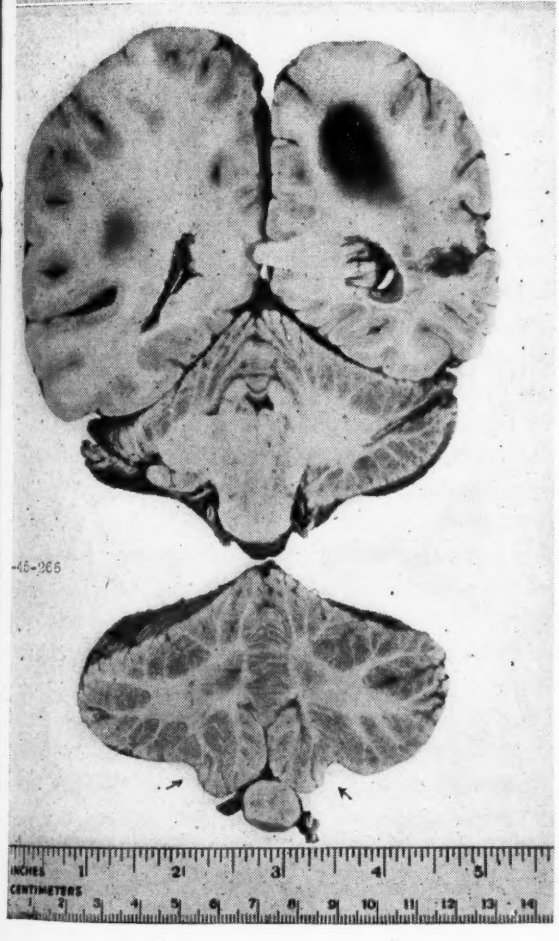
Coronal sections through both hemispheres revealed broadening of the white matter, compression and narrowing of the cortical gray matter and almost complete obliteration of the lateral ventricles (Figure 1, the corpus callosum was severed at autopsy to allow for better penetration of the formalin solution). There was a small area of recent softening transecting the left internal capsule near the head of the caudate nucleus. In the white matter of the left occipital lobe there was a ball hemorrhage (Figure 2) measuring 8 mm. in diameter. There was the scar of an old softening in the right hippocampus and an old cyst 1 cm. in length involving the lateral portion of the left internal capsule and the mesial portion of the globus pallidus (Fig. 1).

The microscopic examination disclosed tremendous swelling of all the constituents of the nervous parenchyma. These changes hardly would have been seen in preparations stained with hematoxylin and eosin; stained by the Bodian and Loyez methods the characteristics of brain swelling were obvious. The majority of the nerve fibers and myelin sheaths were irregularly swollen; numerous fusiform varicosities were found throughout their course. The glia did not escape the process of swelling. In all sections taken from the white matter there was rarefaction of the tissue. The subcortical U-fibers were relatively well preserved. The cortical ribbon was compressed but otherwise was normal.

There were two principal types of vascular abnormalities: arteriolar hyalinization characteristic of hypertensive arteriolopathy, and

congestion and stasis of veins associated with degeneration and atrophy of their wall. Similar venous changes have been described in a large series of patients with hypertensive brain disease associated with ball and massive hemorrhage.[1]





The diffuse involvement of large areas of the brain by acute swelling could account for the elevated cerebrospinal fluid pressure and must be responsible for some of the symptoms of hypertensive brain disease. Clinical manifestations similar to those presented by our patient have been described under the heading of vascular crisis (Pal) or malignant hypertension. Characteristically there is sudden onset of headache, drowsiness, confusion, impaired vision, and usually increased intracranial pressure. The acute clinical manifestations may last for days and then remit, or occasionally may be followed soon by death. The characteristic pathologic finding in all cases is brain swelling. The designation "hypertensive brain swelling" would therefore seem to be more appropriate than that of vascular crisis or malignant hypertension.

Clinical Diagnosis

Hypertensive disease of the brain, retina, heart, and kidney.

Doctor Ferris' Diagnosis

Malignant hypertension.

Chronic pyelonephritis with renal insufficiency.

Diffuse cerebral vascular disease with multiple small intracerebral hemorrhages, including one in the pons.

Diffuse venous lesions.

Brain edema.

Anatomical Diagnosis

Hypertensive brain disease.

Cerebral edema.

Multiple, small, old and new areas of cerebral softening.

Ball hemorrhage, left occipital lobe.

Advanced active and chronic pyelonephritis with marked arteriolonephrosclerosis.

FIG. 1 (above) — FIG. 2 (below)

Fig. 1: Vertical section of the cerebral hemispheres at the level of the thalamus, to illustrate the swelling of the white matter and thinning of the cortical gray matter. A cyst in the left internal capsule and the shrunken first temporal convolution on the left are also depicted, also narrowing of the ventricles.

Fig. 2: Vertical section through both occipital lobes to show the ball hemorrhage on the left; also note the destruction of the gray matter bordering the posterior extent of the Sylvian fissure on the left. Evidence of cerebellar pressure cone is also indicated (arrows).

Generalized arteriolosclerosis.
Cardiac hypertrophy.
Moderate chronic passive congestion.

Discussion

Student: Would you discuss the absence of papilledema in this case?

Dr. Ferris: I cannot be dogmatic about it, but believe it was because the process developed very rapidly. The onset of the crisis was about 1½ hours before admission and he died within 66 hours. In patients who develop hypertensive encephalopathy it takes time for papilledema to occur. I have recently followed one patient in whom the onset of encephalopathy could be timed exactly and it took 48 hours for recognizable papilledema to develop, although increased intracranial pressure of the order of 500 mm., C.S.F., and retinal hemorrhage were present from the beginning of the attack. In this respect the development of papilledema may be no different from peripheral edema; once the conditions for edema are set, it takes time for it to form.*

Dr. Kurt Tschiassny: What was the lesion in the pons?

Dr. I. Mark Scheinker: We did not examine the pons as closely as it should have been done, and it is possible that microscopic lesions may have been overlooked.

Dr. Ferris: I would like to make a few remarks both with regard to the nature of the syndrome of hypertensive encephalopathy and in reference to the significance of the term vasoparalysis. I think we have a good bit of evidence that patients with malignant hypertension have increased vascular sensitivity in that the arterioles go into spasm more readily than do normal blood vessels.

The patient in an encephalopathic phase of hypertension has evidence of severe arterial spasm not only in the optic fundi but also in the kidney and superficial blood vessels of the extremities. Transient episodes of focal cerebral arterial spasm are indicated in hypertension by the transient paralyses and the migraine-like headaches.

In my experience migraine is extremely common in hypertensive patients. I mention migraine at this time because our recent studies of migraine occurring spontaneously[2] and as the result of decompression sickness,[3] show clearly that the initial disturbance in migraine is a focal arteriolar spasm with cerebral hypoxia. Further, in a minority of cases of migraine the circulatory disturbance is diffuse enough to produce transient hemiplegia or even coma, symptoms indistinguishable from those seen in hypertension; to be sure these migrainous patients do not necessarily have hypertension. I mention this to indicate that the basis for hypertensive encephalopathy is a diffuse cerebral arteriolar spasm.

In hypertensive encephalopathy Doctor Scheinker has demonstrated diffuse involvement of cerebral venules which he has termed vasoparalysis, to which is attributed certain of the cerebral signs. It is my feeling that the venous lesions which Doctor Scheinker has described represent one part of the reaction to hypoxia which in the case of hypertensive encephalopathy is primarily due to prolonged and severe spasm of cerebral arterioles.

The phenomenon of vasoparalysis consists of dilatation of venules, stasis of blood in their lumina with separation of cells and plasma and finally, the development of thrombi and degeneration of the vein wall. Various stages of this phenomenon have been frequently described in the peripheral capillaries and venules in conditions associated with diminished blood flow and anoxemia, resulting either from transient arteriolar spasm, shock, or mechanically induced vascular obstruction.

Eichna[4] has noted venous sludging in normal vessels, Swindle[5] has described it in relation to decompression sickness, and Knisley[6] has described it in a number of conditions, related either to diminished cardiac output or to arteriolar spasm. More recently the entire sequence of events has been observed to occur in frost bite[7,8] where the initial disturbance is prolonged arteriolar spasm causing tissue anoxia. Later the arterioles relax, the veins dilate, edema occurs, and the phenomenon of sludging takes place which may

*See Lauber, Hans: "Formation of papilledema," *Arch. Ophthal.*, 13: 733-743, 1935. He demonstrated that if there is a relatively high diastolic venous pressure in the retina in relation to the diastolic arterial pressure, papilledema is not apt to occur even in the face of markedly increased intracranial pressure.

finally result in venous thrombosis.

Lange et al[9] have presented evidence that the process, up to the point of thrombus formation, is reversible and they have been able to prevent gangrene in frost bitten animals by administration of heparin which presumably prevents thrombus formation. I think that the parallelism between the vascular lesion in frost bite and that in hypertensive encephalopathy together with the evidence concerning the nature of migraine suggest that the common initial lesion in the latter two diseases is cerebral arteriolar spasm, diffuse and prolonged in hypertensive encephalopathy, focal and transient in migraine.

The use of cranial decompression as a method of treatment has been suggested.[10] Such therapy was not pertinent in the case discussed today because of the progressive renal insufficiency. In similar cerebral crises which are not associated with renal insufficiency, the process is a reversible one and patients often recover spontaneously from severe attacks of encephalopathy. The use of decompression in this disease is highly debatable and in reviewing a series of cases subjected to this treatment, I was not convinced that the operation did much good. We do occasionally see dramatic improvement after decompression by lumbar drainage of cerebrospinal fluid. Insofar as the increased intracranial pressure is contributing to the cerebral hypoxia a decompression procedure is indicated; however, one must remember that the cerebral edema and high intracranial pressure are secondary phenomena and their relief will not necessarily relax the arteriolar spasm.

One final point concerns the significance of focal neurological signs in hypertensive encephalopathy. On the medical wards we see a great many cases of hypertensive encephalopathy and it is striking how seldom we observe focal signs. Contrariwise on the neurological service focal signs are common. Obviously the nature of the signs makes the decision as to which service the patient is to be assigned. In this case the occurrence of focal signs suggested that the patient had more than cerebral edema and that is why I thought the patient had a small hemorrhage or area of softening in addition to the cerebral edema.

Dr. Scheinker: I believe in this case of diffuse cerebral damage it would be rather difficult to be sure what was responsible for the focal signs. Grossly there was no hemorrhage or softening in the pons, but I can believe that microscopically one may have found a small local lesion.

We are in agreement about the clinical evidence for angiospasm.[11] In pathology we do not see angiospasm, we see the end result which is vasoparalysis. Vasoparalysis possibly is preceded by angiospasm. We are inclined to blame any transient paralysis or dysphasia on angiospasm whether or not the patient is suffering from hypertensive encephalopathy. However, a consistent accompaniment of hypertensive brain disease is generalized swelling, which is not seen in other types of encephalopathy.

Dr. Ferris: Varying degrees of edema follow any prolonged temporary period of arteriolar spasm; this is true in reactive hyperemia after temporary arterial occlusion, in frost bite and in immersion foot. Even in migraine there may well be focal brain swelling secondary to arteriolar spasm and to my knowledge, one such case has been described.[12]

REFERENCES

1 Scheinker, I. M.: "Changes in Cerebral Veins in Hypertensive Brain Disease and Their Relation to Cerebral Hemorrhage," *Arch. Neurol. Psychiat.*, 54: 395-408, 1945.

2 Engel, G. L., Ferris, E. B., and Romano, J.: "Focal Electroencephalographic Changes During Scotoma of Migraine," *Am. J. Med. Sci.*, 209: 650-657, 1945.

3 Engel, G. L., Webb, J. P., Ferris, E. B., Romano, J., Ryder, H., and Blankenhorn, M. A.: "A Migraine-like Syndrome Complicating Decompression Sickness," *War. Med.*, 5: 304-314, 1944.

4 Eichna, L. W.: Personal communication.

5 Swindle, P. F.: Personal communication.

6 Knisely, M. H.: Personal communication.

7 Greene, R.: "Immediate Vascular Changes in True Frost Bite," *J. Path. and Bact.*, 55: 259-267, 1943.

8 Lange, K., and Boyd, L. J.: "Use of Fluorescein to Determine Adequacy of Circulation," *Med. Cl. N. A.*, 26: 943-952, 1942.

9 Lange K., Boyd, L. J., and Loewe, L.: "The Functional Pathology of Frost Bite and the Prevention of Gangrene in Experimental Animals and Humans," *Science*, 102: 151-152, 1945.

10 McIntyre, H. D.: Personal communication.

11 Aring, C. D.: "Vascular Diseases of the Nervous System," *Brain*, 68: 28-55, 1945.

12 Goltman, A. M.: "Mechanism of Migraine," *J. Allergy*, 7: 351-355, 1936.

Control of Grand Mal Epilepsy with Electro-Shock

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Since first reporting the use of electro-shock on epileptic patients in 1942,* we have continued its use on our small epileptic population. Although the New Jersey State Hospital at Trenton does not have as large an epileptic population as the State colony at Skillman has, nevertheless, a certain number of patients who are escape problems or who are subject to violent furors are hospitalized at Trenton for custodial reasons. We are continuing our study of the effect of electro-shock on the small group available.

We began treating these cases in 1941 with the idea of producing a convulsion under controlled conditions and thereby eliminate any spontaneous convulsion. The method of treatment was to produce an epileptiform seizure of the grand mal type with the standard electro-shock apparatus, beginning with three treatments a week. This was continued for five weeks and if the patient had no spontaneous convulsions, the electro-shock was cut to twice a week. After two weeks, if no spontaneous convulsion occurred, it was administered once a week and the patient kept on this regime indefinitely. If convulsions occurred during the weekly or semi-weekly administration, the treatments were returned to the three times a week schedule. No sedative medication was administered and the patients were fed the regular hospital diet.

The original group of patients consisted of 19, of whom 8 were transfers from the Epileptic Colony because of homicidal tendencies. The others were patients admitted to this hospital and diagnosed "Psychosis due to Grand Mal Epilepsy". Of the total, 5 improved sufficiently under treatment to warrant release into the community and 4 of the transfer cases were able to return to that institution and have continued to make a good adjustment there. Of the remainder, 8 are still in residence at this hospital, 2 have died.

During the course of treatment of these 19 patients, one showed no improvement, continuing to have daily seizures during the first series. In this patient however, when the treatment was administered for the third series, the convulsions decreased to approximately one a month. A second patient had convulsions every 3 to 4 days during the first series of treatment but in the second series, had no convulsions during the treatment. All of the other 17 patients had no convulsions while on electric shock and showed a marked improvement in their demeanor.

Due to the shortage of personnel, we were unable, until recently, to keep these patients under close observation. In the past four months however, we have segregated female epileptics and kept a careful watch for any epileptic manifestations. A brief summary of the cases now on treatment follows:

M.B.: White female, age 34. Epileptic seizures for the past 9 years. Prior to receiving electric shock patient was having frequent convulsions and had received dilantin. The first day she was on the Shock Therapy Ward she had 4 grand mal seizures. Electro-shock was then begun and for a period of 4 months there have been no further seizures. For the past 2 months she has been receiving one electric shock a week.

H.M.: White female, age 38. History of epilepsy for the past 5 years. This patient has had no definite grand mal seizures but prior to electro-shock treatment, was subject to frequent furors and her hospital record shows her attacking other patients on an average of once every 2 weeks. She was placed on electro-shock and for 4 months has made an excellent ward adjustment. She is taking a great interest in her personal appearance, is doing excellent work in the Occupational Therapy Shop and her release is being arranged for.

C.S.: White female, age 31. Epilepsy of 17 years duration. For the past year she has been having epileptic seizures at the rate of 3 or 4 a month. During the first month of her treatment with electric shock, she had 3 grand mal seizures. Since that time she has had no convulsions and

*Proceedings of Phila. Psy. Soc., Archives Neurology & Psychiatry, May 1943, Vol. 49, p. 786.

makes a good adjustment on the ward although she shows a marked deterioration.

M.B.: White female, age 28. History of convulsions of 17 years duration. Patient, prior to treatment with electric shock was having numerous convulsions, averaging one a day. She was placed on electro-shock and in the past 4 months has had 4 convulsions, all spaced at weekly intervals. For the past month she has had only one convulsion.

I.C.: White female, age 47. History of convulsions for the past 20 years. Prior to treatment with electric shock she was averaging 3 convulsions a week. She was placed on electric shock and has had no convulsions for the past 4 months.

J.C.: White female, age 26. History of epilepsy for the past 4 years. This patient was observed in several convulsions 2 years ago. She was placed on electro-shock and improved and has had no further convulsions but has developed considerable irritability and emotional instability. A second course of electro-shock was instituted 4 months ago and patient has had 1 grand mal convulsion 2 months ago. The treatment is being continued once a week and no further convulsions have been noted. This patient has improved to the point where she is now being released from the hospital.

Only one case of status epilepticus was treated: Patient was a white male, age 42, who had been subject to epilepsy for 30 years. He was admitted to this hospital in 1938 and had a seizure approximately every other day until September 2, 1941. On that day he had 5 seizures. These continued to increase daily until on September 8th, he had 13 seizures and continued at this rate until the 12th. He was then given his first electric shock and during the 24 hour period following this, he had 10 seizures. Another electro-shock was given and in the next 24 hours he had one seizure. A course of 15 electric shocks was instituted and patient remained free from convulsions for a period of 4 months. On January 11, 1942 he had one convulsion and another course of electric shock was instituted. This kept him free from convulsions for a period of one month, when, because of the patient's insistence, the family withdrew permission for treatment. Since that time, patient has continued to have a seizure approximately once a week.

With the elimination or decrease of spontaneous convulsions, we have noted a marked decrease in the number of furors, a marked improvement in patient's general demeanor with less evidence of deterioration.

While the number of cases reported are few, they do suggest that in patients having frequent epileptic seizures or equivalents and where large amounts of sedative medication are required, a controlled convulsion produced by electric shock, will be of benefit in limiting spontaneous convulsions and in helping the patient make a better social adjustment.

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